Shaver ?/rau
1998 /rpy

=> d 1-6 ibib ab kwic

L1 ANSWER 1 OF 6 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 2002:433997 SCISEARCH
THE GENUINE ARTICLE: 552ZR
TITLE: Chromosome instability in lymphocytes: A potential indicator of predisposition to oral premalignant lesions
AUTHOR: Wu X F (Reprint); Lippman S M; Lee J J; Zhu Y; Wei Q V; Thomas M; Hong W K; Spitz M R
CORPORATE SOURCE: Univ Texas, MD Anderson Canc Ctr, Dept Epidemiol, Box 189,
1515 Holcombe Blvd, Houston, TX 77030 USA (Reprint); Univ Texas, MD Anderson Canc Ctr, Dept Epidemiol, Houston, TX 77030 USA; Univ Texas, MD Anderson Canc Ctr, Dept Clin Canc Prevent, Houston, TX 77030 USA; Univ Texas, MD Anderson Canc Ctr, Dept Biomath, Houston, TX 77030 USA; Univ Texas, MD Anderson Canc Ctr, Dept Thorac Head & Neck Med Oncol, Houston, TX 77030 USA
COUNTRY OF AUTHOR: USA
SOURCE: CANCER RESEARCH, (15 MAY 2002) Vol. 62, No. 10, pp. 2813-2818.
Publisher: AMER ASSOC CANCER RESEARCH, PO BOX 11806, BIRMINGHAM, AL 35202 USA.
ISSN: 0008-5472.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 56

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Oral premalignant lesions (OPLs) are related to tobacco use and mark individuals at high risk for oral cancer development. Increased mutagen sensitivity as measured by an in vitro mutagen challenge assay has been shown to be a risk factor for upper aerodigestive tract cancers. In this case control study, we used two assays with mutagens relevant to tobacco exposure (benzo[a]pyrene diol epoxide (BPDE) and bleomycin) to see whether sensitivity to these mutagens could be used as biomarkers for assessing risk of premalignant lesions. Furthermore, we evaluated whether 3p21.3 is a molecular target of BPDE damage in lymphocytes of patients with OPLs. There were 82 patients with OPLs and 89 healthy controls frequency matched to the cases on age, sex, ethnicity, and smoking status. These subjects' lymphocytes were treated in two separate experiments with either 2 muM BPDE for 24 h or 0.03 units/ml bleomycin for 5 h, and the frequency of induced chromatid breakage in Giemsa-stained preparations was determined. BPDE-induced 3p21.3 aberrations were scored by fluorescent in situ hybridization technique in 1000 interphases/sample. We found that the mean BPDE-induced chromatid breaks per cell were higher in cases than controls (1.05 +/- 0.40 and 0.55 +/- 0.27, respectively; P < 0.01). Similar results were evident with bleomycin-induced chromatid breaks per cell (0.78 +/- 0.37 and 0.57 +/- 0.31, respectively; P < 0.01). After adjusting for age, sex, ethnicity, and smoking status, significantly elevated odds ratios (95% confidence interval) for OPL risk were noted for BPDE sensitivity [12.96 (5.51, 30.46)] and bleomycin sensitivity [3.33 (1.64, 6.77)]. When subjects were categorized into quartiles of the number of breaks per cell, a dose response was observed for both assays. The adjusted odds ratios for subjects with increasing numbers of breaks per cell in quartiles were

2.34, 9.14, and 54.04 for BPDE sensitivity and 1.92, 3.33, and 7.15 for bleomycin sensitivity, respectively. Subjects sensitive to both mutagens had a 50-fold increased risk for OPLs. In addition, there were significantly more BPDE-induced chromosome aberrations at the 3p21.3 locus in cases (51.13/1000) than in controls (40.93/1000; P < 0.0001). However, no such difference was observed for 3q13, a control locus. BPDE-induced 3p21.3 aberrations were associated with an elevated risk for OPLs of 6.08 (2.57, 14.4). The degree of BPDE sensitivity at 3p21.3 and risk for OPLs increased in a dose-dependent manner. In summary, BDPE sensitivity and bleomycin sensitivity appear to be individually. and jointly associated with elevated risk of OPLs. Furthermore, 3p21.3 may be a molecular target of BPDE in OPLs. This is the first study to examine mutagen sensitivity in a premalignant condition. The next step is to correlate these findings in surrogate (lymphocyte) tissue with molecular events in the target tissue.

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)
SHRIVER S P	1998	406	9	MUTAT RES-GENOMICS <--

L1 ANSWER 2 OF 6 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 2002:334041 SCISEARCH
THE GENUINE ARTICLE: 540ZR
TITLE: Autoantibody against ribosomal protein L14 in patients with systemic lupus erythematosus
AUTHOR: Hasegawa H (Reprint); Uchiumi T; Sato T; Saito A; Nakano M; Gejyo F
CORPORATE SOURCE: Univ Tennessee, Ctr Hlth Sci, Dept Mol Sci, 858 Madison Ave, Suite G01, Memphis, TN 38163 USA (Reprint); Niigata Univ, Sch Med, Dept Med 2, Niigata, Japan; Shinshu Univ, Fac Text Sci & Technol, Inst High Polymer Res, Ueda, Nagano 386, Japan; Sato Med Clin, Joetsu, Japan
COUNTRY OF AUTHOR: USA; Japan
SOURCE: CLINICAL AND EXPERIMENTAL RHEUMATOLOGY, (MAR-APR 2002) Vol. 20, No. 2, pp. 139-144.
Publisher: CLINICAL & EXPER RHEUMATOLOGY, VIA SANTA MARIA 31, 56126 PISA, ITALY.
ISSN: 0392-856X.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 29

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Objective To isolate a specific antibody, against ribosomal protein L14 and to assess the relationship of this antibody with some of the clinical features in patients with systemic lupus erythematosus (SLE).

Methods We screened the sera of SLE patients by immunoblotting analysis using rat total ribosomal proteins as antigen to determine whether sera had antibody activity against ribosomal proteins other than the P, S10, and L12 proteins. The sera from 2 patients had antibody activity against a 30-kDa ribosomal protein. This antigenic protein was identified to be ribosomal protein L14 by two-dimensional gel electrophoresis and immunoblotting, so the antibody against L14 was tested by immunoblotting analysis using glutathione-S-transferase fusion human-L14 protein (GST-L14) as the antigen. We examined sera from 126 patients with SLE, and as controls sera from 67 patients with dermatomlyositis and polymyositis

(DM/PM), 71 patients with systemic sclerosis (SSc), and 74 healthy donors.

Results Antibody, activity, against GST-L14 was detected in 7 out of 126 SLE, but not in any of the DM/PM, PSS, or healthy, controls.

Conclusion Antibody against ribosomal protein L14 was specifically detected in sera from patients with SLE. Although this antibody, activity was not so prevalent in the patients with SLE, it might be one of the useful tools for diagnosis of SLE.

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	
SHRIVER S P	1998	406	9	MUTAT RES-GENOMICS	<--

L1 ANSWER 3 OF 6 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 2002:48887 SCISEARCH
THE GENUINE ARTICLE: 507UW
TITLE: Differential expression of genes coding for ribosomal proteins in different human tissues
AUTHOR: Bortoluzzi S; d'Alessi F; Romualdi C; Danieli G A (Reprint)
CORPORATE SOURCE: Univ Padua, Dept Biol, Via G Colombo 3, I-35131 Padua, Italy (Reprint); Univ Padua, Dept Biol, I-35131 Padua, Italy; Univ Padua, CRIBI Biotechnol Ctr, I-35131 Padua, Italy
COUNTRY OF AUTHOR: Italy
SOURCE: BIOINFORMATICS, (DEC 2001) Vol. 17, No. 12, pp. 1152-1157.
Publisher: OXFORD UNIV PRESS, GREAT CLARENDON ST, OXFORD OX2 6DP, ENGLAND.
ISSN: 1367-4803.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 23
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Motivation: To perform a computational and statistical study on a large set of gene expression data pertaining six adult human tissues (brain, liver, skeletal muscle, ovary, retina and uterus) for analyzing the expression of ribosomal protein genes.

Results: Unexpectedly, in each of the considered tissues large variations in the expression of ribosomal protein genes were observed. Moreover, when comparing the expression levels of 89 ribosomal protein genes in six different tissues, 13 genes appeared differentially expressed among tissues.

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	
SHRIVER S P	1998	406	9	MUTAT RES-GENOMICS	<--

L1 ANSWER 4 OF 6 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 2001:445926 SCISEARCH
THE GENUINE ARTICLE: 434XF
TITLE: Inhibition of protein synthesis by the T cell receptor-inducible human TDAG51 gene product
AUTHOR: Hinz T (Reprint); Flindt S; Marx A; Janssen O; Kabelitz D
CORPORATE SOURCE: Paul Ehrlich Inst, Dept Immunol, Paul Ehrlich Stasse 51-59, D-63225 Langen, Germany (Reprint); Paul Ehrlich Inst, Dept Immunol, D-63225 Langen, Germany; Univ Kiel, Inst Immunol, D-24105 Kiel, Germany

COUNTRY OF AUTHOR: Germany
SOURCE: CELLULAR SIGNALLING, (MAY 2001) Vol. 13, No. 5, pp. 345-352.
Publisher: ELSEVIER SCIENCE INC, 655 AVENUE OF THE AMERICAS, NEW YORK, NY 10010 USA.
ISSN: 0898-6568.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 32

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The T cell death associated gene 51 (TDAG51) was shown to be required for T cell receptor (TCR)-dependent induction of Fas/Apol/ CD95 expression in a murine T cell hybridoma. Despite the absence of a nuclear localization sequence and a nucleic acid binding domain, it was suggested to be localized in the nucleus and to function as a transcription factor regulating Fas-expression. However, we demonstrate that the human (h)TDAG51 protein is localized in the cytoplasm and the nucleoli, suggesting a role in ribosome biogenesis and;or translation regulation. Indeed, it strongly inhibited translation of a luciferase mRNA in a reticulocyte translational extract. Furthermore, cotransfection of hTDAG51 and the luciferase gene into 293T cells resulted in a strong inhibition of luciferase mRNA translation. Our findings were further strengthened by isolating in a yeast two-hybrid screen three proteins which are involved in the regulation of translation. Pie speculate that hTDAG51 couples TCR signaling to inhibition of protein biosynthesis in activated T lymphocytes. (C) 2001 Elsevier Science Inc. All rights reserved.

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)
SHRIVER S P	1998	406	9	MUTAT RES-GENOMICS <--

L1 ANSWER 5 OF 6 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 2001:414817 SCISEARCH
THE GENUINE ARTICLE: 431VN
TITLE: LUCA-15 suppresses CD95-mediated apoptosis in Jurkat T cells
AUTHOR: Sutherland L C (Reprint); Lerman M; Williams G T; Miller B A
CORPORATE SOURCE: Sigfried & Janet Weis Ctr Res, Geisinger Clin, Henry Hood Res Program, 100 N Acad Ave, Danville, PA 17822 USA (Reprint); Sigfried & Janet Weis Ctr Res, Geisinger Clin, Henry Hood Res Program, Danville, PA 17822 USA; Penn State Univ, Milton S Hershey Med Ctr, Coll Med, Dept Pediat, Div Hematol Oncol, Hershey, PA 17033 USA; NCI, Immunobiol Lab, FCRDC, Frederick, MD 21702 USA; Univ Keele, Sch Life Sci, Keele ST5 5BG, Staffs, England
COUNTRY OF AUTHOR: USA; England
SOURCE: ONCOGENE, (10 MAY 2001) Vol. 20, No. 21, pp. 2713-2719.
Publisher: NATURE PUBLISHING GROUP, HOUNDMILLS, BASINGSTOKE RG21 6XS, HAMPSHIRE, ENGLAND.
ISSN: 0950-9232.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English

REFERENCE COUNT: 50

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The candidate tumour suppressor gene, LUCA-15, maps to the lung cancer tumour suppressor locus 3p21.3. Overexpression of an alternative RNA splice variant of LUCA-15 has been shown to retard human Jurkat T cell proliferation and to accelerate CD95-mediated apoptosis, An antisense cDNA to the 3'-UTR of this splice variant was able to suppress CD95-mediated apoptosis, Here, we report that overexpression of LUCA-15 itself suppresses CD95-mediated apoptosis in Jurkat cells. This suppression occurs Drier to the final execution stage of the CD95 signalling pathway, and is associated with upregulation of the apoptosis inhibitory protein Bcl-2, LUCA-15 overexpression is also able to inhibit apoptosis induced by the protein kinase inhibitor staurosporine, but is not able to significantly suppress apoptosis mediated by the topoisomerase II inhibitor etoposide, These findings suggest that LUCA-15 is a selective inhibitor of cell death, and confirm the importance of the LUCA-15 genetic locus in the control of apoptosis.

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)
SHRIVER S P	1998	406	9	MUTAT RES-GENOMICS <--

L1 ANSWER 6 OF 6 SCISEARCH COPYRIGHT 2002 ISI (R)

ACCESSION NUMBER: 2000:546972 SCISEARCH

THE GENUINE ARTICLE: 333WN

TITLE: Genetic aberrations in oral or head and neck squamous cell carcinoma 2: chromosomal aberrations

AUTHOR: Scully C (Reprint); Field J K; Tanzawa H

CORPORATE SOURCE: UNIV LONDON, UNIV COLL LONDON, EASTMAN DENT INST ORAL HLTH CARE SCI, 256 GRAYS INN RD, LONDON WC1X 8LD, ENGLAND (Reprint); UNIV LIVERPOOL, MOL GENET & ONCOL GRP, LIVERPOOL L69 3BX, MERSEYSIDE, ENGLAND; ROY CASTLE INT CTR LUNG CANC RES, LIVERPOOL, MERSEYSIDE, ENGLAND; CHIBA UNIV, DEPT ORAL SURG, CHUO KU, CHIBA 2608670, JAPAN

COUNTRY OF AUTHOR: ENGLAND; JAPAN

SOURCE: ORAL ONCOLOGY, (JUL 2000) Vol. 36, No. 4, pp. 311-327. Publisher: PERGAMON-ELSEVIER SCIENCE LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD OX5 1GB, ENGLAND. ISSN: 0964-1955.

DOCUMENT TYPE: General Review; Journal

FILE SEGMENT: CLIN

LANGUAGE: English

REFERENCE COUNT: 288

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Carcinogenesis is the result of a series of genetic mutations resulting in unregulated growth of a clone of cells and the development of malignant lesion that is largely monoclonal though, with the evolution of further genetic changes, there develops a degree of heterogeneity in the tumour. DNA technology, especially allelic imbalance (loss of heterozygosity) studies have identified chromosomal changes in oral carcinoma and head and

neck squamous cell carcinoma (SCCHN), suggestive of the involvement of tumour suppressor genes (TSGs), particularly in chromosomes 3, 9, 11 and 17. The regions most commonly identified have included 3p, especially 3p14.2 (FHIT); 3p24, and 3p21.3. where the TSGs involved are as yet unidentified; 9p21 where p16(INK4A/MTS-1] is the main target TSG; and 17p13 where p53 is the major target TSC. Over-expression of oncogenes, genes mainly involved in cell signalling, especially those on chromosome 11 (PRAD-1 in particular) and 17 (H-ras) and mutations in DNA repair genes, have also been implicated in the carcinogenesis of SCCHN.

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	
SHRIVER S P	1998	406	9	MUTAT RES-GENOMICS	<--

L4 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:123271 CAPLUS
DOCUMENT NUMBER: 136:178945
TITLE: Detection and diagnosis of smoking related **cancers** with gene probes specific to chromosome 3 and 10
INVENTOR(S): Katz, Ruth; Jiang, Feng
PATENT ASSIGNEE(S): Board of Regents, the University of Texas System, USA
SOURCE: PCT Int. Appl., 79 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002012563	A2	20020214	WO 2001-US24718	20010806

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2000-222811P P 20000804

AB The invention provides gene probes specific for regions of chromosome 3 (3p21.3) and chromosome 10 (10q22) as tools for the diagnosis and prognosis of smoking related **cancers** such as non-small cell lung **cancer** (NSCLC). In particular, the invention discloses these probes, including gene probes contg. **RPL14**, CD39L3, PMGM, GC20 and PTEN, can be used with fluorescence in situ hybridization (FISH), and used to stratify smokers into high and low risk groups, as well as det. a patients susceptibility to the development of smoking related **cancers**. The invention also provides methods for identifying a subject at high risk for the development, recurrence, or metastasis of **cancer**.

L4 ANSWER 2 OF 2 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 1999116779 MEDLINE
DOCUMENT NUMBER: 99116779 PubMed ID: 9920051
TITLE: Trinucleotide repeat length variation in the human ribosomal protein L14 gene (**RPL14**): localization to 3p21.3 and loss of heterozygosity in lung and oral **cancers**.
AUTHOR: Shriver S P; Shriver M D; Tirpak D L; Bloch L M; Hunt J D; Ferrell R E; Siegfried J M
CORPORATE SOURCE: Department of Pharmacology and University of Pittsburgh Cancer Institute, University of Pittsburgh, PA 15261, USA..
sshriver@vms.cis.pitt.edu
CONTRACT NUMBER: P20 CA58235 (NCI)
SOURCE: MUTATION RESEARCH, (1998 Nov) 406 (1) 9-23.
Journal code: 0400763. ISSN: 0027-5107.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199902
ENTRY DATE: Entered STN: 19990223
Last Updated on STN: 19990223
Entered Medline: 19990205

AB Chromosome 3p is consistently deleted in lung **cancer**, oral squamous cell **carcinoma**, and renal cell **carcinoma**, and is believed to contain several tumor suppressor genes. We have shown a role for chromosome 3 in tumor suppression by microcell-mediated chromosome transfer experiments. We have isolated a gene that is located at 3p21.3 within the smallest region of deletion overlap in lung tumors and is the human homolog of the ribosomal protein L14 gene (**RPL14**). The **RPL14** sequence contains a highly polymorphic trinucleotide repeat array which encodes a variable-length polyalanine tract. Genotype analysis of **RPL14** shows that this locus is 68% heterozygous in the normal population, compared with 25% in non-small cell lung **cancer** (NSCLC) cell lines (p = 0.008). Cell cultures derived from normal bronchial epithelium show a 65% level of heterozygosity, reflecting that of the normal population. Squamous cell **carcinoma** of the head and neck (SCCHN), which has the same risk factors as lung **cancer** and is hypothesized to have a similar etiology, demonstrates 54% loss of heterozygosity at the RNA level, suggesting that transcriptional loss may be a primary mechanism of **RPL14** alteration in SCCHN. In addition, **RPL14** shows significant differences in allele frequency distribution in ethnically-defined populations, making this sequence a useful marker for the study of ethnicity-adjusted lung **cancer** risk.

ANSWER 2 OF 2 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 1998:174147 SCISEARCH
THE GENUINE ARTICLE: YY270
TITLE: Triplet repeat-containing **ribosomal protein L14** gene in immortalized human endothelial cell line (t-HUE4)
AUTHOR: Tanaka M (Reprint); Tanaka T; Harata M; Suzuki T; Mitsui Y
CORPORATE SOURCE: NATL INST BIOSCI & HUMAN TECHNOL, 1-1 HIGASHI, IBARAKI, OSAKA 305, JAPAN (Reprint); TOKAI UNIV, SCH MED, DIV HOST DEV MECHANISM, KANAGAWA 25911, JAPAN; GIFU UNIV, DEPT BIOTECHNOL, GIFU 5011193, JAPAN
COUNTRY OF AUTHOR: JAPAN
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (13 FEB 1998) Vol. 243, No. 2, pp. 531-537.
Publisher: ACADEMIC PRESS INC JNL-COMP SUBSCRIPTIONS, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495.
ISSN: 0006-291X.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 40

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB A cDNA encoding human 60S ribosomal subunit protein L14 (hRL14) was isolated from a human immortal endothelial cell line, t-HUE4. This cell line was established via a series of cell lines cultured in a serum-free and a protein-free medium, and a directional cDNA library has been constructed and screened in search for the genes modulating protein synthesis machinery in cell proliferation. A putative full-length clone with an open reading frame of 220 amino acids; predicted molecular weight of 23.6 kDa. A significant identity for hRL14 was observed with rat RL14 (85% identity), with exception of COOH-terminal region, but not with any eukaryote amino acid sequences so far deposited to database. The typical features of ribosomal proteins were observed in hRL14, as seen in nuclear targeting sequences necessary for the transport from cytoplasm to nucleolus, a bZIP like (basic region-leucine zipper) element for the binding to rRNA, and the internal repeat sequences; the pentapeptide QKA(A/S)X The COOH-terminal region of the transcripts contained fifteen triplet repeats (GCT; alanine) at nucleotide 465 to 509, which is significantly expanded compared to the rat RL14. However, the repeat number was all the same among the normal human endothelial cell line and the cell Lines established in the course of t-HUE4 establishment. A single band with about 800 bases was identified by Northern blot analysis without tissue specificity. This GCT repeat was found to be one of the longest uninterrupted repeats in a coding sequence, which were associated with the highest degree of polymorphism. (C) 1998 Academic Press.

L9 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:396923 CAPLUS
DOCUMENT NUMBER: 135:15131
TITLE: Protein and cDNA of a human **ribosomal protein L14**.22 and therapeutic use thereof
INVENTOR(S): Mao, Yumin; Xie, Yi
PATENT ASSIGNEE(S): Bioroad Gene Development Ltd. Shanghai, Peop. Rep. China
SOURCE: PCT Int. Appl., 30 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Chinese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001038389	A1	20010531	WO 2000-CN471	20001120
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CN 1297905	A	20010606	CN 1999-124098	19991124

PRIORITY APPLN. INFO.: CN 1999-124098 A 19991124

AB The invention provides cDNA sequences for a novel human **ribosomal protein L14**.22 cloned from placenta brain, and its protein sequences which have sequence homol. to Drosophila melanogaster counterpart. The invention also relates to constructing **ribosomal protein L14**.22 gene expression vectors to prep. recombinant **ribosomal protein L14**.22 protein using prokaryote or eukaryote cells. Methods of expressing and prepg. recombinant **ribosomal protein L14**.22 protein and its antibody are described. Methods of using **ribosomal protein L14**.22 gene or protein products for the treatment of various kinds of diseases, such as **cancer**, blood diseases, HIV infection, immune diseases and inflammation are also disclosed.